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Pan-immune inflammation value as a determinant of coronary collateral circulation in patients with chronic coronary syndrome and chronic total occlusion

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ABSTRACT

Aims: This study aimed to investigate the association between coronary collateral circulation (CCC) pan-immune-inflammation value (PIV) in patients with chronic coronary syndrome (CCS) and chronic total occlusion (CTO).

Methods: The study included 297 patients with CCS who underwent coronary angiography and had CTO in at least one major coronary artery. Patients with CTO were categorized into two groups based on Rentrop grading: group 1 (grades 2–3, well-developed CCC) and group 2 (grades 0–1, poor CCC).

Results: Patients with poor CCC had higher WBC levels (p=0.04), neutrophil counts (p=0.001), platelet counts (p=0.023), and median PIV values (p=0.003) compared to patients with well-developed CCC. Logistic regression identified PIV (per hundred units) as an independent predictor of poor CCC (OR=1.10, 95% [CI=1.02–1.23], p=0.044). Receiver operating characteristic (ROC) analysis demonstrated that a cut-off value of 168 for PIV predicted poor CCC slightly better compared to other markers, with 94.6% sensitivity and 22.8% specificity (area under the curve=0.601, 95% CI=0.543–0.657, p=0.002).

Conclusion: These findings indicate that PIV may serve as an independent predictor of CCC development.

Keywords: Coronary collateral circulation, pan-immune-inflammation value, chronic coronary syndrome, chronic total occlusion

INTRODUCTION

Coronary artery disease (CAD) is characterized by the buildup of atherosclerotic plaques and is a leading cause of death and disability globally.[1] Coronary collateral circulation (CCC) refers to the small blood vessels that connect different coronary arteries or segments of the same artery. Under normal physiological conditions, these collateral vessels are typically very small and play a minimal role in coronary blood flow. However, in response to chronic or repeated myocardial ischemia, these collateral vessels gradually enlarge and develop into a functional network of collateral circulation.[2] The role of CCC is to function as an alternative blood vessel, thereby ensuring the area beyond the blocked segment of the coronary artery receives a sufficient blood supply. This may assist in mitigating myocardial ischemia. Research has indicated that the presence of well-developed CCC in patients with CTO can be associated with enhanced survival and improved overall prognosis.[3,4] However, it should be noted that CCC formation can vary significantly among patients. The current methods for assessing CCC formation, such as the collateral flow index and intracoronary electrocardiogram, are both costly and complex. Consequently, there is a need to identify a simple, cost-effective biomarker for evaluating CCC formation.

The precise mechanisms underlying CCC formation remain unclear. However, research has shown that inflammation can impede the growth of collateral formation by affecting the development of new blood vessels. Studies have indicated that inflammatory biomarkers derived from complete blood counts, such as the platelet-to-lymphocyte ratio (PLR) and the neutrophil-to-lymphocyte ratio (NLR), are linked to CCC formation and can be useful for evaluating it.[5,6]

There are very few studies in the literature that specifically examine the role of PIV in predicting CCC formation. Therefore, this study aims to examine the relationship between PIV and CCC formation in patients with CTO and to evaluate whether PIV is a more effective predictor of CCC formation compared to other inflammatory biomarkers.

METHODS

Ethics

The study was conducted with the permission of Başakşehir Çam and Sakura Hospital Scientific Researches Ethics Committee No. 1 (Date: 16.07.2024, Decision No: KAEK/10.07.2024.109). All procedures were carried out in

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accordance with the ethical rules and the principles of the Declaration of Helsinki.

Study Population

A total of 297 patients over 18 years of age, diagnosed with CCS according to the criteria recommended by the European Society of Cardiology and who had CTO in ≥1 major coronary artery and underwent coronary angiography (CAG) between March 2021 and January 2023, were included in this retrospective study conducted at the Cardiology Department of Başakşehir Çam and Sakura City Hospital. CTO were defined as lesions persisting for more than three months, characterized by either a complete interruption of antegrade blood flow on angiography or minimal contrast penetration through the lesion without opacification of the distal vessel.[7,8] CAG is mostly indicated because of typical chest pain or results of noninvasive stress tests suggesting myocardial ischemia (positive stress test result and/or ischemia on myocardial perfusion scintigraphy). Patients were excluded from the study if they had previous coronary artery bypass graft surgery, history of acute coronary syndrome within 3 months, presence of left main coronary stenosis of 50% or more, history of previous percutaneous coronary intervention, left ventricular ejection fraction (LVEF) <40%, severe valvular disease, hematologic disease, malignancy, severe renal (estimated glomerular filtration rate <30 ml/min/1.73 m^2 calculated using the modification of diet in renal disease formula) or hepatic disease, ongoing infection or chronic inflammatory disease, and autoimmune disease.

A detailed medical history was obtained from all patients, along with a physical examination, complete blood count, and serum biochemistry tests. Classical cardiovascular risk factors such as age, gender, diabetes mellitus (DM), hypertension (HT), dyslipidemia, and smoking were evaluated. All patients underwent a transthoracic echocardiographic examination. The definitions of DM, HT, and dyslipidemia followed established criteria.[9]

Laboratory Analysis

Venous blood samples were collected from the antecubital region of all patients into tripotassium EDTA-based anticoagulant tubes before CAG. Samples were collected in the morning after a 20-min rest period followed by a 12-h fasting period. Hemoglobin, platelet count, white blood cell (WBC) count, neutrophils, lymphocytes, glucose levels, lipid profile, and other routine biochemical tests were analyzed using an autoanalyzer (Roche Diagnostic Modular Systems). Tripotassium EDTA-based anticoagulant blood samples were stored at 4°C and analyzed with a Sysmex K-1000 autoanalyzer (Sysmex) within 30 min of collection.

PIV was calculated as neutrophil count multiplied by platelet count multiplied by monocyte count divided by lymphocyte count (or multiplying the monocyte count with SII).

Transthoracic Echocardiography (TTE)

Transthoracic echocardiography was performed for each patient before CAG. M-mode and 2D ECHO were performed in the left lateral decubitus position using a 3.25 probe from the Vivid 5 ECHO echocardiography device, according to the American Society of Echocardiography criteria.[10] Parasternal short-long axis images and apical 4 and 2 chamber views, which are standard echocardiography positions, were used for measurements. LVEF was calculated using the Modified Simpson method.[11]

Coronary Angiography Methods

CAG was performed using the standard Judkins technique using either the femoral or radial access, depending on the operator's preference. CAG image evaluations were performed by two experienced interventional cardiologists. CCC was assessed using the Rentrop classification: grade 0=no filling in any collateral vessels; grade 1=filling in the lateral branches of the artery supplying the epicardial segment; grade 2=partial filling of the epicardial artery via collateral vessels; and grade 3=complete filling of the epicardial artery by a collateral vessel.[12] Rentrop grade 0 was considered to indicate no CCC development. In addition, Rentrop grades 2 and 3 were classified as satisfactory CCC development (group 1), while Rentrop grades 0 and 1 were classified as poor CCC development (group 2). If more than one coronary collateral vessel was observed on CAG, classification was made according to the vessel with the highest Rentrop grade. Intra-observer and inter-observer variability for CCC assessment were found to be 2% and 3%, respectively.

Statistical Analysis

All data tests were conducted using the Statistical Package for the Social Sciences 25.0 for Windows (SPSS Inc., Chicago, IL, USA). Continuous variables were tested for normality using the Kolmogorov-Smirnov or Shapiro-Wilk test. Data were presented as mean±standard deviation (SD) for normally distributed variables or median [interquartile range-IQR_{25-75}] for non-normally distributed variables. Categorical variables were expressed as frequencies and percentages.

Comparisons between the two groups (group 1: Rentrop 2–3; group 2: Rentrop 0–1) were conducted using the following tests; The independent samples t-test was applied for normally distributed continuous variables. The Mann-Whitney U test was used for non-normally distributed continuous variables. The Chi-square test was utilized for categorical variables. A receiver operating characteristic (ROC) curve analysis was performed to evaluate the predictive ability of PIV for the determination of poor CCC. The area under the curve (AUC) and corresponding 95% confidence interval (CI) were calculated. The optimal cutoff value for PIV (per hundred units) was determined based on Youden's index, and sensitivity and specificity values were reported. To identify independent predictors of poor CCC (Rentrop 0–1), logistic regression analysis was conducted. Variables with a p-value <0.05 in univariable analysis were included in the multivariable model. The Youden index was utilised in order to ascertain a cut-off point for PIV in the assessment of collateral circulation. Results were expressed as odds ratios (OR) with 95% CI. A p-value <0.05 was considered statistically significant for all analyses.

RESULTS

The demographic variables, including gender distribution, mean age, prevalence of HT, DM, and smoking status, were

comparable between well-developed and poor coronary circulation groups, with no statistically significant differences observed (Table 1). The mean age was 64.5±10.5 years in group 1 (well-developed) and 63.3±10.6 years in group 2 (poor). Similarly, the distribution of HT (p=0.686), DM (p=0.591), and smoking status (p=0.96) was balanced between both groups. Several laboratory parameters demonstrated significant differences between the two groups. WBC counts in group 2 had significantly higher WBC levels compared to group 1 (9.5±3.3 vs. 8.8±3.1, p=0.04). High-density lipoprotein cholesterol (HDL-C) levels were significantly lower in group 2 (38.8±8.4 mg/dl) compared to group 1 (42.0±11.7 mg/dl; p=0.007). The median PIV values were higher in group 2 (347.5 [260.3–666.9]) compared to group 1 (331.2 [175.4–491.1]; p=0.003). Neutrophil counts in group 2 exhibited a higher compared to group 1 (5.5 [4.6–6.9] vs. 4.9 [4.0–6.0]; p=0.001). Similarly, platelet counts in group 2 had higher (268.8±82.5 vs. 247.2±80.3; p=0.023). Table 1 details the clinical characteristics and laboratory results of the study population by CCC.

Multivariable logistic regression analysis was performed to determine independent predictors of poor CCC development. The results revealed that PIV (OR=1.10, 95% CI=1.02–1.23, p=0.044, per hundred units) and urea (OR=0.98, 95% CI=0.97-0.99, p=0.013) were a significant independent predictor (Table 2). The variable importance analysis, incorporating the parameters from the univariable analysis, indicated that PIV (per hundred units) was the variable with the most significant contribution to the model in determining poor CCC (Figure 1).

The predictive capacity of PIV for the determination of poor CCC was assessed using ROC analysis, which yielded an AUC of 0.601 (95% CI=0.543-0.657, p=0.002). A cut-off value of >168

Table 2. Multivariable logistic regression analysis of determinants of poor collateral circulation development

Variable	Odds ratio	95% confidence interval	p*
HDL-C	0.98	0.95-1.00	0.094
PIV (per hundred units)	1.10	1.02-1.23	0.044
Urea	0.98	0.97-0.99	0.013
LVEF (%)	0.98	0.96-1.01	0.122
Neutrophil	1.03	0.89-1.19	0.714
Platelet	1.00	0.99-1.01	0.648
Monocyte	1.51	0.48-4.77	0.486
Hyperlipidemia	1.41	0.86-2.31	0.171

HD-CL: High-density lipoprotein cholesterol, LVEF: Leftt ventricular ejection fraction, PIV: Pan-immune- inflammation value. *p value <0.05 was considered significant

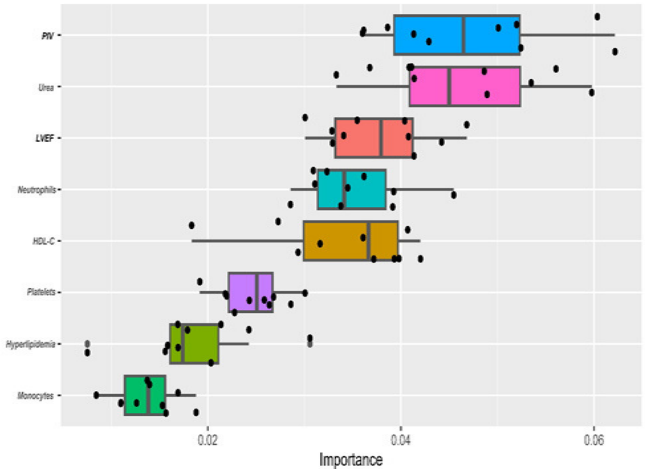

Figure 1. Variable importance plot

PIV: Pan-immune-inflammation value, LVEF: Left ventricular ejection fraction, HDL-C: High-density lipoprotein cholesterol

Table 1. Clinical and laboratory characteristics of the study population by coronary collateral circulation

		Coronary collateral circulation		
Characteristic	**Levels**	**Group 1 (n=149) (well-developed)**	**Group 2 (n=148) (poor)**	**p***
Gender	Male	111 (74.5%)	116 (78.4%)	0.515
Age, years	Mean (SD)	64.5 (10.5)	63.3 (10.6)	0.320
HT, n (%)	Yes	54 (36.2%)	58 (39.2%)	0.686
DM, n (%)	Yes	66 (44.3%)	60 (40.5%)	0.591
Smoking, n (%)	Yes	63 (42.3%)	64 (43.2%)	0.960
Hyperlipidemia, n (%)	Yes	75 (50.3)	63 (42.6)	0.180
LVEF (%)	Mean (SD)	55.5 (9.6)	53.4 (11.2)	0.078
Hgb (g/dl)	Mean (SD)	13.7 (1.9)	13.8 (2.2)	0.911
Plt (10^3/μL)	Mean (SD)	247.2 (80.3)	268.8 (82.5)	0.023
Monocyte (10^3/μL)	Median (IQR)	0.53 (0.41-0.70)	0.60 (0.50-0.79)	0.006
Lymphocytes (10^3/μL)	Median (IQR)	2.1 (1.7-2.7)	2.2 (1.7-2.9)	0.521
Urea (mg/dl)	Median (IQR)	36 (26-50)	33 (28-44)	0.209
HDL-C (mg/dl)	Mean (SD)	42.0 (11.7)	38.8 (8.4)	0.007
WBC (10^3/μL)	Mean (SD)	8.8 (3.1)	9.5 (3.3)	0.040
NEU (10^3/μL)	Median (IQR)	4.9 (4.0–6.0)	5.5 (4.6–6.9)	0.001
PIV	Median (IQR)	331.2 (175.4–491.1)	347.5 (260.3–666.9)	0.003

Values are n (%), median (interquartile range [IQR]), or mean±standard deviation. p value was calculated using an independent samples t-test or the Mann-Whitney U-test for continuous variables and a Chi-squared test or the Fisher's exact test for categorical variables, as appropriate. SD: Standard deviation, HT: Hypertension, DM: Diabetes mellitus; Hgb: Hemoglobin, HDL-C: High-density, LVEF: Leftt ventricular ejection fraction, Lipoprotein cholesterol, WBC: White blood cell count, NEU: Neutrophil count, Plt: Platelet count, IQR, Interquartile Range; PIV, Pan-immune-inflammation value, *p value <0.05 was considered significant.

was determined, with 94.6% sensitivity and 22.8% specificity, indicating a modest discriminatory ability (Figure 2).

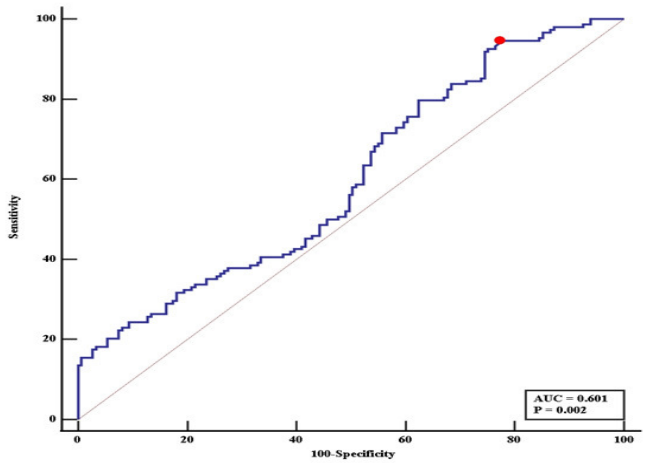

Figure 2. The analysis of receiver operating characteristic curves in determining the presence of poor collateral circulation

DISCUSSION

Our findings in this study highlight the significant association between high PIV and impaired collateral circulation in CCS patients with CTO, suggesting that systemic inflammation may play an important role in the development of coronary collaterals.

CCC serves as an important compensatory mechanism to maintain myocardial perfusion in the presence of CAD, particularly in the case of significant stenosis or occlusion. Well-developed CCC has been shown to enhance ventricular function and limit infarct size. Furthermore, it may reduce the incidence of cardiovascular events, lower mortality risk and improve the prognosis of patients with CTO.[13,14]

Chronic inflammation impairs the development of CCC by contributing to endothelial dysfunction, partly through the increased production of reactive oxygen species.[15] Platelets and various subtypes of leukocytes such as neutrophils, lymphocytes, and monocytes serve as key effector cells in the inflammatory response. Platelets, in particular, store both pro- and anti-angiogenic factors that regulate neovascularization in response to ischemia. Neutrophils contribute to vascular endothelial damage by releasing large amounts of reactive oxygen species, along with inflammatory mediators and proteolytic enzymes. Elevated neutrophil counts have been associated with impaired collateral vessel development. While monocytes are known to promote angiogenesis, it is primarily tissue-resident monocytes not circulating ones hat play a pivotal role in the process of arteriogenesis.[16] Monocytes also contribute to local ischemia and endothelial dysfunction. Evidence from previous studies indicates that patients with poorly developed CCC exhibit higher monocyte counts compared to those with well-developed CCC.[5] Lymphocyte counts tend to decline in response to systemic inflammation. This reduction may impair the formation of CCC by decreasing levels of vascular endothelial growth factor and other pro-angiogenic mediators, as well as by limiting vascular infiltration necessary for collateral development.[17]

Building on the previous findings, several inflammatory biomarkers derived from peripheral immune cells have been identified as potential diagnostic markers for CCC formation in patients with CTO. Among these, NLR and PLR have been shown to be associated with CCC formation and may serve as predictors for its development in CTO patients.[5,6]

However, PLR and NLR alone do not fully capture the complex immune and inflammatory landscape, as they only assess the counts of two types of immune-inflammatory cells. Recently, the PIV has emerged as a more comprehensive immunoinflammatory biomarker that better reflects the overall immune and inflammatory status. The PIV includes all types of blood inflammatory cells (e.g., neutrophils, lymphocytes, monocytes, and platelets). Recent research has demonstrated that PIV outperforms NLR and PLR in predicting the prognosis of STEMI patients.[18]

In the study conducted by Çetinkaya et al.[19] PIV was found to be significantly associated with higher Syntax and CAD severity in non-STEMI patients. In another study conducted by Keleşoğlu et al.[20] in 354 patients, they found that stable CAD and high CRP/albumin ratio could be an independent predictor of poor CCC. These two studies, similar to our study, showed that inflammatory markers increased in severe vascular occlusion and associated poor CCC.

Similar to our study, in a study conducted by Zhang et al.[21] on 1150 CTO patients, PIV was found to be associated with the development of CCC. In another study by Yilmaz et al.[22] including 663 patients with CCS, PIV was found to be an independent predictor of the development of CCC, similar to our study.

Consistent with previous studies, our findings demonstrated that patients with poor CCC exhibited significantly elevated levels of inflammatory markers, including WBC, neutrophil count, and PIV.[21,22] The PIV offers a more comprehensive evaluation of systemic inflammation by integrating the contributions of both platelet activity and immune cell activation. Notably, our analysis revealed that high PIV levels were independently associated with poor CCC, similar to the literature.

Wu et al.[23] demonstrated the relationship between PIV and long-term prognosis in patients with HT, indicating that the inflammatory markers incorporated in PIV could have broader clinical applications in predicting outcomes beyond cardiovascular disease. These findings further suggest that PIV could be a valuable tool in various disease settings.

Limitations

This study has several limitations. It was a single-center, retrospective study with a relatively small patient population. PIV was measured only at the time of hospitalization. The study results would have been stronger if inflammatory markers such as PLR, NLR, and systemic immune index were measured together with PIV. In addition, the cross-sectional study design prevents the establishment of causality between systemic inflammation and the development of CCC. Further prospective studies, especially multicenter studies, are needed to confirm these findings and to investigate the underlying

mechanisms by which inflammation affects collateral vessel formation.

CONCLUSION

Our study suggests that PIV could be a significant determinant of poor CCC among patients with CCS and CTO, underscoring the importance of systemic inflammation in collateral vessel development. The PIV may serve as a useful biomarker in identifying patients at risk for impaired collateral circulation and could guide therapeutic interventions aimed at improving myocardial perfusion in CAD patients.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was conducted with the permission of Başakşehir Çam and Sakura Hospital Scientific Researches Ethics Committee No. 1 (Date: 16.07.2024, Decision No: KAEK/10.07.2024.109).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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